	Initial values			One month follow-up		
Mean	SA	UA	p value	SA	UA	p value
TPA (pg/ml)	13.91	22.50	0.006	11.76	13.67	0.292
vWF (Um/ml)	2265	1802	0.020	1864	1654	0.333
PAI-1 (pg/ml)	27.54	21.49	0.231	29.82	31.72	0.770

Mean values of plasma TPA and vWF were significantly higher in UA patients than in SA patients ($p = 0.006$ and 0.020, respectively). There were no significant differences between plasma levels of PAI-1 in the two groups ($p = 0.231$). One month post procedure, there was a decrease in plasma levels of TPA and vWF in the UA patients. There were no longer any significant differences in the plasma levels of TPA and vWF between UA and SA patients. These data suggest that plasma levels of TPA and vWF may correlate with instability of atheromatous plaques and that their decrease post procedure might reflect plaque stabilization.

902-2 Incidence of Prodromal Symptoms in Patients with Acute Myocardial Infarction in a Defined Geographic Population

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The incidence of prodromal symptoms has significant implications since intervention before the onset of acute myocardial infarction may be accomplished with beneficial effect. In Olmsted County, Minnesota, we looked at all patients presenting to the hospital with acute myocardial infarction between 1988 and 1995. Of these patients, 335 (Group 1; 34.9 percent) had pain only with their acute myocardial infarction. Their first symptom of coronary artery disease was chest pain associated with the myocardial infarction. An additional 436 patients (Group 2; 45.4 percent) had unstable angina in the two months prior to their myocardial infarction. Unstable angina was defined as new onset angina, crescendo angina, rest angina, or prolonged angina lasting more than 20 minutes. An additional 45 patients (Group 3; 4.7 percent) had stable chronic angina before their myocardial infarction and 144 patients (Group 4; 15 percent) had no pain with their acute myocardial infarction and presented to the hospital for other symptoms. Of the patients with pain only during a myocardial infarction (Group 1), the in hospital mortality was 3.9 percent. Of patients with prodromal unstable angina symptoms (Group 2), the mortality was 11.5 percent ($p < 0.05$ compared to Group 1). In patients with stable angina pre-myocardial infarction (Group 3), the mortality was 13.3 percent. In Group 4, the mortality was 23 percent. Total in hospital mortality for all patients was 10.6 percent.

Conclusion: 1) Forty-five percent of patients with acute myocardial infarction from a geographic based population have prodromal symptoms in the two months prior to their myocardial infarction. 2) Patients with prodromal symptoms are a high risk population with increased mortality when compared to patients that have no prior symptoms.

902-3 Effect of the Patency of Infarct-Related Artery on Vagal Reflexes

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In post myocardial infarction (MI) patients (pts) the restoration of antegrade flow in the infarct related artery (IRA) significantly improves survival. We tested the hypothesis that patency of IRA may enhance vagal reflexes, a factor known to affect electrical stability of the infarcted myocardium. Analysis of angiographic data was performed in 324 of 419 post MI pts enrolled in a multicenter prospective study within 6 weeks after the index MI (Post-Infarction Risk Stratification = PIRS). Pts underwent baroreflex sensitivity assessment by the Phenylepiphrine method and determination of heart rate variability (HRV, standard deviation of RR intervals (SDNN) assessed from 24 h Holters). IRA patency was documented in 226 pts (70%) and in the remaining 98 pts (30%) the artery remained occluded.

Results:

	Pts	BRS < 3 ms/mmHg	BRS (M ± SD)	HRV < 75 ms	HRV (M ± SD)
TIMI 0-II	98	23%	8.5 ± 6.1	26%	99 ± 32
TIMI III	226	18%	9.5 ± 7.3	17%	109 ± 36
p-value		0.04	0.05	0.05	0.01

M ± SD = mean ± standard deviation

Conclusion: The presence of an open IRA is associated with a higher HRV and BRS. These data correlate with the improvement of survival after MI, if restoration of antegrade flow in the IRA is possible.

902-4 Effects of Residual Flow on the Recovery from Myocardial Stunning: A Myocardial Contrast Echocardiographic Study

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Myocardial contrast echo (MCE) was performed to assess the salutary effects of pre-reperfusion residual flow (RF) on the recovery from myocardial stunning. The size of occluded bed, an area supplied by infarct-related artery, was determined by comparing pre- and post- reperfusion MCE in 46 pts with first acute myocardial infarction. MCE defect (no reflow) was observed after reperfusion in 10 pts. The pts without MCE no reflow were subdivided into 2 groups based on the ratio of the area perfused by RF to that of the occluded bed: those with the ratio of >10% (RF(+)) and the others (RF(−)). The wall motion score (0, normal, to 4, dyskinetic) was obtained in the convalescent stage.

Results: The changes in wall motion score were as follows:

Day	Good reflow		No reflow
	RF(+) (n = 14)	RF(−) (n = 22)	n = 10
Just after:	2.6 ± 1.1*	3.1 ± 0.8	3.5 ± 0.5
3-day:	2.3 ± 1.2*#	3.1 ± 0.8	3.5 ± 0.5
1-week:	1.9 ± 1.1*#	2.9 ± 1.0	3.7 ± 0.5
2-week:	1.6 ± 1.2*#	2.8 ± 1.1	3.7 ± 0.5
4-week:	1.3 ± 1.0*#	2.7 ± 1.1*	3.7 ± 0.5
6-month:	1.0 ± 0.7*#	2.4 ± 1.2*	3.7 ± 0.5

(mean ± SD: *$p < 0.05$ vs. no reflow, #$p < 0.05$ vs. RF (−))

(1) Wall motion of the infarct area after 3-day was better in pts with RF than in pts without RF. (2) LV function in long-term improved remarkably in pts with good reflow and RF(+), improved mildly in pts with good reflow and RF(−), but did not improve in pts with no reflow. *Conclusion:* The presence of residual flow within the infarct area before reperfusion results in not only good myocardial salvage but also rapid functional recovery from myocardial stunning.

902-5 The Ischemic Threshold of Hibernating Myocardium Is Higher When Hibernation Is Chronic as Compared to Short-term

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To investigate whether the ischemic threshold differs between short-term and chronic hibernating myocardium with the same degree of hypoperfusion, we studied 29 pigs with myocardial hibernation characterized by severe regional LV dysfunction subtending a severe LAD stenosis and recovered myocardial lactate production. The short-term group (n = 16) was studied from 90 min to 24 hrs and the chronic group at 7 days. Dobutamine stress echocardiography was performed with incremental doses from 2.5 to 30 μg/kg/min to determine the ischemic threshold, defined as deterioration of regional wall thickening or regional myocardial lactate production. *Results:* Resting coronary flow was 0.49 ± 0.21 ml/100 g myocardium, a 41 ± 6% reduction in short-term, and 0.52 ± 0.17 ml/100 g myocardium, a 40 ± 7% reduction in chronic hibernation (p = NS). Baseline anterior regional wall thickening was also similar in the 2 groups: 11 ± 10% for short-term vs. 10 ± 7% for chronic hibernation (p = NS). In the chronic group, regional wall thickening increased initially and then decreased to 10 ± 10% at a dobutamine dose of 20 ± 5 μg/kg/min. In contrast, deterioration of wall thickening, to 4 ± 8%, was observed at a lower dobutamine dose, 12 ± 7 μg/kg/min ($p < 0.05$) in the short-term group. Lactate production was lower in the chronic group at each dobutamine dose and at the maximal dose ($p < 0.01$ for each). The ischemic threshold was reached at 7 ± 5 mins in the short-term group and at 12 ± 3 mins in the chronic hibernation group ($p < 0.05$). *Conclusions:* The ischemic threshold of short-term hibernating myocardium is low. With more chronic myocardial hibernation, the ischemic threshold increases through an as yet undefined mechanism This finding is relevant to the management of acute coronary syndromes with hibernation.

902-6 Non-Culprit Artery Blood Flow in Acute Coronary Syndromes is Related to Culprit Artery Blood Flow: A RESTORE substudy

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The relationship between flow in culprit and non-culprit arteries was analyzed in pooled data from pts. with acute coronary syndromes undergoing PTCA